

What is claimed is:

1. A method of preventing or treating a disease of a mammal, wherein at least one symptom of the disease is mediated at least in part by the binding of an effector molecule to a DC-SIGN receptor of the mammal to be treated, wherein the method comprises administering to the mammal an amount of a DC-SIGN modulator sufficient to substantially modulate the binding of the effector molecule to the DC-SIGN receptor to thereby prevent or treat the disease.

2. A method of preventing or treating a disease of a mammal, wherein at least one symptom of the disease is mediated at least in part by the binding of an effector molecule to a DC-SIGN receptor of the mammal to be treated, wherein the method comprises administering to the mammal an amount of a DC-SIGN blocker sufficient to substantially inhibit the binding of the effector molecule to the DC-SIGN receptor to thereby prevent or treat the disease.

3. The method of claim 2, wherein the DC-SIGN blocker is a blocking derivative of the effector molecule.

4. The method of claim 2, wherein the DC-SIGN blocker is an antibody.

5. The method of claim 4, wherein the antibody specifically binds DC-SIGN.

FINNEGAN
HENDERSON
FARABOW
GARRETT &
DUNNER LLP

1300 I Street, NW
Washington, DC 20005
202.408.4000
Fax 202.408.4400
www.finnegan.com

6. The method of claim 4, wherein the antibody specifically binds the effector molecule.

7. The method of claim 2, wherein the DC-SIGN blocker is a mannosylated molecule that binds to a DC-SIGN receptor.

8. The method of claim 7, wherein the mannosylated molecule is mannan.

9. A method of preventing or treating a viral infection of a mammal, wherein the viral infection is mediated at least in part by the binding of a viral effector molecule to a DC-SIGN receptor of the mammal to be treated, wherein the method comprises administering to the mammal an amount of a DC-SIGN modulator sufficient to substantially modulate the binding of the viral effector molecule to the DC-SIGN receptor to thereby prevent or treat the viral infection.

10. A method of preventing or treating a viral infection of a mammal, wherein the viral infection is mediated at least in part by the binding of a viral effector molecule to a DC-SIGN receptor of the mammal to be treated, wherein the method comprises administering to the mammal an amount of a DC-SIGN blocker sufficient to substantially inhibit the binding of the viral effector molecule to the DC-SIGN receptor to thereby prevent or treat the viral infection.

FINNEGAN
HENDERSON
FARABOW
GARRETT &
DUNNER LLP

1300 I Street, NW
Washington, DC 20005
202.408.4000
Fax 202.408.4400
www.finnegan.com

11. The method of claim 10, wherein the viral effector molecule is a molecular constituent of the viral envelope.

12. The method of claim 11, wherein the molecular constituent of the viral envelope is an envelope glycoprotein.

13. The method of claim 10, wherein the DC-SIGN blocker comprises a binding moiety of the viral effector molecule.

14. The method of claim 12, wherein the DC-SIGN blocker comprises a binding moiety of the envelope glycoprotein.

15. The method of claim 10, wherein the DC-SIGN blocker is an antibody.

16. The method of 15, wherein the antibody is a monoclonal antibody.

17. The method of claim 16, wherein the mammal is a human and the monoclonal antibody is humanized.

18. The method of claim 15, wherein the antibody specifically binds DC-SIGN.

19. The method of claim 16, wherein the monoclonal antibody is Mab
1B10.2.6.

20. The method of claim 15, wherein the antibody specifically binds the viral
effector molecule.

21. The method of claim 20, wherein the antibody specifically binds the
binding moiety of the viral effector molecule.

22. The method of claim 10, wherein the DC-SIGN blocker is a mannosylated
molecule that binds to a DC-SIGN receptor.

23. The method of claim 22, wherein the mannosylated molecule is mannan.

24. The method of claim 10, wherein the viral infection is a CMV infection
and the viral effector molecule is a CMV effector molecule.

25. The method of claim 24, wherein the mammal is a human.

26. The method of claim 24, wherein the CMV effector molecule is a
molecular constituent of the CMV envelope.

27. The method of claim 26, wherein the molecular constituent of the CMV envelope is a CMV envelope glycoprotein.

28. The method of claim 27, wherein the CMV envelope glycoprotein is CMV envelope glycoprotein B.

29. The method of claim 24, wherein the DC-SIGN blocker comprises a binding moiety of the CMV effector molecule.

30. The method of claim 28, wherein the DC-SIGN blocker comprises a binding moiety of the CMV envelope glycoprotein B.

31. The method of claim 30, wherein the DC-SIGN blocker is a recombinantly produced protein.

32. The method of claim 24, wherein the DC-SIGN blocker is an antibody.

33. The method of 32, wherein the antibody is a monoclonal antibody.

34. The method of claim 33, wherein the mammal is a human and the monoclonal antibody is humanized.

35. The method of claim 32, wherein the antibody specifically binds DC-SIGN.

36. The method of claim 33, wherein the monoclonal antibody is Mab 1B10.2.6.

37. The method of claim 32, wherein the antibody specifically binds the CMV effector molecule.

38. The method of claim 37, wherein the CMV effector molecule is CMV envelope glycoprotein B.

39. A method of preventing or treating an Ebola, HIV or SIV infection of a human or a simian, wherein the method comprises administering to the human or simian an amount of a DC-SIGN modulator sufficient to substantially modulate the binding of Ebola, HIV or SIV to the DC-SIGN receptor present on dendritic cells of the human or simian to thereby prevent or treat the Ebola, HIV or SIV infection.

40. A method of preventing or treating an Ebola, HIV or SIV infection of a human or a simian, wherein the method comprises administering to the human or simian an amount of a DC-SIGN blocker sufficient to substantially inhibit the binding

of Ebola, HIV or SIV to the DC-SIGN receptor present on dendritic cells of the human or simian to thereby prevent or treat the Ebola, HIV or SIV infection.

41. The method of claim 40, wherein the DC-SIGN blocker comprises a binding moiety of the CMV envelope glycoprotein B.

42. The method of claim 40, wherein an HIV infection of a human is prevented or treated.

43. A method of preventing or treating inflammation in a mammal caused by specific binding of ICAM-3 present on T cells of the mammal with DC-SIGN receptor present on dendritic cells of the mammal, wherein the method comprises administering to the mammal an amount of a DC-SIGN modulator sufficient to substantially modulate the binding of ICAM-3 present on T cells of the mammal with DC-SIGN receptor present on dendritic cells of the mammal to thereby prevent or treat inflammation.

44. A method of preventing or treating inflammation in a mammal caused by specific binding of ICAM-3 present on T cells of the mammal with DC-SIGN receptor present on dendritic cells of the mammal, wherein the method comprises administering to the mammal an amount of a DC-SIGN blocker sufficient to substantially inhibit the binding of ICAM-3 present on T cells of the mammal with

DC-SIGN receptor present on dendritic cells of the mammal to thereby prevent or treat inflammation.

45. The method of claim 44, wherein the DC-SIGN blocker comprises a binding moiety of the CMV envelope glycoprotein B.

46. The method of claim 44, wherein the mammal is a human.

47. A pharmaceutical composition comprising:

- a) A DC-SIGN modulator, and
- b) at least one pharmaceutically acceptable excipient;

wherein the DC-SIGN blocker is present in the composition at an achievable therapeutic concentration.

48. A pharmaceutical composition comprising:

- a) A DC-SIGN blocker, and
- b) at least one pharmaceutically acceptable excipient;

wherein the DC-SIGN blocker is present in the composition at an achievable therapeutic concentration.

49. The pharmaceutical composition of claim 48, wherein the DC-SIGN blocker is a derivative of a viral effector molecule.

50. The pharmaceutical composition of claim 48, wherein the DC-SIGN blocker comprises the binding moiety of a CMV effector molecule.

51. The pharmaceutical composition of claim 50, wherein the CMV effector molecule is CMV envelope glycoprotein B.

52. The pharmaceutical composition of claim 48, wherein the DC-SIGN blocker is an antibody.

53. The pharmaceutical composition of claim 52, wherein the antibody is a monoclonal antibody.

54. The pharmaceutical composition of claim 53, wherein the monoclonal antibody is humanized.

55. The pharmaceutical composition of claim 52, wherein the antibody specifically binds DC-SIGN.

FINNEGAN
HENDERSON
FARABOW
GARRETT &
DUNNER LLP

1300 I Street, NW
Washington, DC 20005
202.408.4000
Fax 202.408.4400
www.finnegan.com

56. The pharmaceutical composition of claim 53, wherein the monoclonal antibody is Mab 1B10.2.6.

57. The pharmaceutical composition of claim 52, wherein the antibody specifically binds the viral effector molecule.

58. The pharmaceutical composition of claim 57, wherein the antibody specifically binds the binding moiety of the viral effector molecule.

59. A method of identifying a DC-SIGN modulator, wherein the method comprises:

a) determining a baseline binding value by:

- i. providing cultured cells comprising a DC-SIGN receptor;
- ii. exposing the cultured cells to a marked viral effector molecule binding moiety for a period of time sufficient to allow binding equilibrium to be reached; and
- iii. determining the extent of binding of the marked viral effector molecule binding moiety to the cultured cells to thereby determine a baseline binding value;

b) determining a test substance binding value by:

- i. providing cultured cells comprising a DC-SIGN receptor;
 - ii. exposing the cultured cells to a marked viral effector molecule binding moiety in the presence of a test substance for a period of time sufficient to allow binding equilibrium to be reached; and
 - iii. determining the extent of binding of the marked viral effector molecule binding moiety to the cultured cells to thereby determine a test substance binding value; and
- c) determining a test substance binding modulation value for the test substance by dividing the test substance binding value by the baseline binding value,

wherein a test substance binding modulation value representing an about 95% inhibition of binding of the viral effector molecule to dendritic cells by the test substance, indicates that the test substance is a substance that substantially modulates the binding of a viral effector molecule to the DC-SIGN receptor.

60. A method of identifying a DC-SIGN blocker, wherein the method comprises:

- a) determining a baseline binding value by:
 - i. providing cultured cells comprising a DC-SIGN receptor;

- ii. exposing the cultured cells to a marked viral effector molecule binding moiety for a period of time sufficient to allow binding equilibrium to be reached; and
 - iii. determining the extent of binding of the marked viral effector molecule binding moiety to the cultured cells to thereby determine a baseline binding value;
- b) determining a test substance binding value by:
- i. providing cultured cells comprising a DC-SIGN receptor;
 - ii. exposing the cultured cells to a marked viral effector molecule binding moiety in the presence of a test substance for a period of time sufficient to allow binding equilibrium to be reached; and
 - iii. determining the extent of binding of the marked viral effector molecule binding moiety to the cultured cells to thereby determine a test substance binding value; and
- c) determining a test substance binding inhibition value for the test substance by dividing the test substance binding value by the baseline binding value,

wherein a test substance binding inhibition value representing an about 95% inhibition of binding of the viral effector molecule to dendritic cells by the test substance, indicates that the test substance is a substance that substantially inhibits the binding of a viral effector molecule to the DC-SIGN receptor.

61. The method of claim 60 wherein the cultured cells are DC.

62. The method of claim 60, wherein the cultured cells are THP-1 cells.

63. The method of claim 60, wherein the viral effector molecule is a CMV effector molecule.

64. The method of claim 63, wherein the CMV effector molecule is CMV envelope glycoprotein B.

65. An isolated DC-SIGN blocker identified by the method of claim 60.

66. A method of targeting a subject molecule to a cell expressing a DC-SIGN receptor by exposing the cell to a targeting complex, wherein the targeting complex comprises a subject molecule and a DC-SIGN blocker, wherein the exposure is under conditions which allow the DC-SIGN blocker to bind to DC-SIGN on the cell

expressing the DC-SIGN receptor, thereby targeting the subject molecule to the cell expressing a DC-SIGN receptor.

67. The method of claim 66, wherein the DC-SIGN blocker is an antibody.

68. The method of claim 67, wherein the antibody is a monoclonal antibody.

69. The method of claim 66, wherein the subject molecule is a protein.

70. The method of claim 66, wherein the subject molecule is an antibody.

71. The method of claim 66, wherein the subject molecule is labeled.

72. The method of claim 66, wherein the exposure occurs *in vivo*.

73. The method of claim 66, wherein the exposure occurs *in vitro*.

74. An isolated antibody, wherein the isolated antibody specifically binds DC-SIGN.

75. An isolated antibody according to claim 74, wherein the antibody is a DC-SIGN modulator.

76. An isolated antibody according to claim 74, wherein the antibody is a DC-SIGN blocker.

77. An isolated monoclonal antibody, wherein the isolated monoclonal antibody specifically binds DC-SIGN.

78. An isolated monoclonal antibody according to claim 77, wherein the monoclonal antibody is a DC-SIGN modulator.

79. An isolated monoclonal antibody according to claim 77, wherein the monoclonal antibody is a DC-SIGN blocker.

80. An isolated monoclonal antibody according to claim 79, wherein the monoclonal antibody is Mab 1B10.2.6, produced by hybridoma 1B10.2.6, deposited at the C.N.C.M. on November 7, 2002, under the accession number I-2951.